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20may03 13:37:15 User208669 Session D2297.1
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File 155:MEDLINE(R) 1966-2003/May W2
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*File 155: Medline has been reloaded and accession numbers have changed. Please see HELP NEWS 155.

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S1 0 SHVEM
S2 295 HVEM
S3 145201 SOLUBLE OR VARIANT
S4 14 S2 AND S3
S5 21 AU=BUSFIELD S?
S6 0 SHVEM?
S7 93583 SOLUBLE
S8 12 S2 AND S7
S9 1 S7 AND S5
S10 0 HVEM2
S11 0 HVEM2DS
S12 31 HERPES? AND S2
S13 75 HVE? AND HERPES?
S14 247214 SOLUBLE OR DOMAIN OR DOMAINS
S15 39 S13 AND S14
S16 27 S13 AND NECTIN
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DIALOG(R)File 155:MEDLINE(R)
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09118869 20417756 PMID: 10961879
Cloning, characterization, and functional studies of human and mouse glycoprotein VI: a platelet-specific collagen receptor from the immunoglobulin superfamily.
Jandrot-Perrus M; Busfield S; Lagrue A H; Xiong X; Debili N; Chickering T ; Le Couedic J P; Goodearl A; Dussault B; Fraser C; Vainchenker W; Villeval J L
Institut National de la Sante et de la Recherche Medicale (INSERM) E9907, Faculte Xavier Bichat, Paris, France.
Blood (UNITED STATES) Sep 1 2000, 96 (5) p1798-807, ISSN 0006-4971 Journal Code: 7603509
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Injuries to the vessel wall and subsequent exposure of collagen from the subendothelial matrix result in thrombus formation. In physiological conditions, the platelet plug limits blood loss. However, in pathologic conditions, such as rupture of atherosclerotic plaques, platelet-collagen interactions are associated with cardiovascular and cerebral vascular diseases. Platelet glycoprotein VI (GPVI) plays a crucial role in collagen-induced activation and aggregation of platelets, and people who are deficient in GPVI suffer from bleeding disorders. Based on the fact that GPVI is coupled to the Fc receptor (FcR)-gamma chain and thus should share homology with the FcR chains, the genes encoding human and mouse GPVI were identified. They belong to the immunoglobulin (Ig) superfamily and share 64% homology at the protein level. Functional evidence demonstrating the identity of the recombinant protein with GPVI was shown by binding to its natural ligand collagen; binding to convulxin (Cvx), a GPVI-specific ligand from snake venom; binding of anti-GPVI IgG isolated from a patient; and association to the FcR-gamma chain. The study also demonstrated that the soluble protein blocks Cvx and collagen-induced platelet aggregation and that GPVI expression is restricted to megakaryocytes and platelets. Finally, human GPVI was mapped to chromosome 19, long arm, region 1, band 3 (19q13), in the same region as multiple members of the Ig superfamily. This work offers the opportunity to explore the involvement of GPVI in thrombotic disease, to develop alternative antithrombotic compounds, and to characterize the mechanism involved in GPVI genetic deficiencies. (Blood. 2000;96:1798-1807)
Record Date Created: 20001018
Record Date Completed: 20001018
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12/7/3
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.
14335344 22353842 PMID: 12466117
Differential expression of LIGHT and its receptors in human placental villi and amniochorion membranes.
Gill Ryan M; Ni Jian; Hunt Joan S; et al
Department of Pathology and Laboratory Medicine, University of Kansas Medical Center, 3901 Rainbow Boulevard, Kansas City, KS 66160-7400, USA.
American journal of pathology (United States) Dec 2002, 161 (6) p2011-7, ISSN 0002-9440 Journal Code: 0370502
Contract/Grant No.: HD 24212; HD; NICHD; HD 33994; HD; NICHD; +
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

mRNA encoding LIGHT (homologous to lymphotoxins, exhibits inducible expression, competes with herpes simplex virus glycoprotein D for HVEM, a receptor expressed by T lymphocytes), a member of the tumor necrosis factor superfamily of ligands, as well as mRNAs encoding LIGHT receptors [HVEM, LTbetaR, and TR6 (DcR3)] are present in placentas and cytotrophoblast cells at term. To establish translation of these messages and determine directions for functional studies, term placentas, amniochorion membranes, and purified cytotrophoblast cells were evaluated by immunoblotting and immunohistochemistry. Ligand and receptor proteins were identified in lysates from all three sources although the soluble receptor, TR6, was scarce in placentas and all receptors were in low abundance in cytotrophoblast cells. These results were confirmed and cell type-specific expression was documented by immunohistochemistry. Ligand and receptor proteins were differentially expressed according to cell type. For example, HVEM was identified on syncytiotrophoblast but not in villous mesenchymal cells; amnion epithelial cells were positive for all proteins whereas chorion membrane cytotrophoblasts exhibited none. Because LIGHT is a powerful cytokine that can alter gene expression and promote apoptosis, these experiments suggest that ligand-receptor interactions may critically influence structural and functional aspects of human placentas through as yet undefined autocrine/paracrine pathways.

Record Date Created: 20021205

Record Date Completed: 20030116

12/7/7

DIALOG(R)File 155:MEDLINE(R)

11814276 99253915 PMID: 10318773

A newly identified member of tumor necrosis factor receptor superfamily (TR6) suppresses LIGHT-mediated apoptosis.

Yu K Y; Kwon B; Ni J; Zhai Y; Ebner R; Kwon B S

Department of Microbiology and Immunology and Walther Oncology Center, Indiana University School of Medicine and the Walther Cancer Institute, Indianapolis, Indiana 46202, USA.

Journal of biological chemistry (UNITED STATES) May 14 1999, 274 (20) p13733-6, ISSN 0021-9258 Journal Code: 2985121R

Contract/Grant No.: AI28125; AI; NIAID; DE12156; DE; NIDCR

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

TR6 (decoy receptor 3 (DcR3)) is a new member of the tumor necrosis factor receptor (TNFR) family. TR6 mRNA is expressed in lung tissues and colon adenocarcinoma, SW480. In addition, the expression of TR6 mRNA was shown in the endothelial cell line and induced by phorbol 12-myristate 13-acetate/ionomycin in Jurkat T leukemia cells. The open reading frame of TR6 encodes 300 amino acids with a 29-residue signal sequence but no transmembrane region. Using histidine-tagged recombinant TR6, we screened soluble forms of TNF-ligand proteins with immunoprecipitation. Here, we demonstrate that TR6 specifically binds two cellular ligands, LIGHT (herpes virus entry mediator (HVEM)-L) and Fas ligand (FasL/CD95L). These bindings were confirmed with HEK 293 EBNA cells transfected with LIGHT cDNA by flow cytometry. TR6 inhibited LIGHT-induced cytotoxicity in HT29 cells. It has been shown that LIGHT triggers apoptosis of various tumor cells including HT29 cells that express both lymphotoxin beta receptor (LTbetaR) and HVEM/TR2 receptors. Our data suggest that TR6 inhibits the interactions of LIGHT with HVEM/TR2 and LTbetaR, thereby suppressing LIGHT- mediated HT29 cell death. Thus, TR6 may play a regulatory role for suppressing in FasL- and LIGHT-mediated cell death.

Record Date Created: 19990617

Record Date Completed: 19990617

12/7/20

DIALOG(R)File 155:MEDLINE(R)

10131802 22110092 PMID: 12115617

The complementation of lymphotoxin deficiency with LIGHT, a newly discovered TNF family member, for the restoration of secondary lymphoid structure and function.

Wang Jing; Foster Amy; Chin Robert; Yu Ping; Sun Yonglian; Wang Yang; Pfeffer Klaus; Fu Yang-Xin

Department of Pathology and Committee on Immunology, The University of Chicago, Chicago, IL 60637, USA.

European journal of immunology (Germany) Jul 2002, 32 (7) p1969-79, ISSN 0014-2980 Journal Code: 1273201

Contract/Grant No.: DK58897; DK; NIDDK; HD37104; HD; NICHD

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Highly organized lymphoid structures provide the intricate microenvironment essential for the mediation of the effective immune responses. Compared with lymphotoxin beta knockout mice (LTbeta-/-), LTbeta receptor knockout (LTbetaR-/-) mice present with more severely disorganized splenic structures, suggesting the potential involvement of another ligand. LIGHT, a newly identified TNF family member, is a costimulatory molecule for T cells and binds to LTbetaR and herpes virus entry mediator (HVEM) in vitro. Here, we show that the complementation of LTalpha-/- mice with a LIGHT transgene (LIGHT Tg/LTalpha-/-) leads to the restoration of secondary lymphoid tissue chemokine and T/B cell zone segregation. LIGHT Tg/LTalpha-/- mice also preserve dendritic cells, follicular dendritic cell networks, and germinal centers, though not the marginal zone. Consequently, IgG responses to soluble, but not particulate, antigens are restored, confirming the role of primary follicle and marginal zone in the responses

to soluble and particulate antigens. The failure of the LIGHT transgene to rescue the defective splenic structures in LTbetaR-/- mice demonstrates that LIGHT can interact with LTbetaR in vivo. More severely disorganized splenic structures developed after blockade of endogenous LIGHT in LTbeta-/- mice. These findings uncover the potential interaction between LIGHT and one of its receptors, LTbetaR, in supporting even in the absence of LT the development and maintenance of lymphoid microenvironment.
Record Date Created: 20020712
Record Date Completed: 20020813

12/7/23
DIALOG(R)File 155:MEDLINE(R)

09973502 21898416 PMID: 11901205
Modulation of LIGHT-HVEM costimulation prolongs cardiac allograft survival.
Ye Qunrui; Fraser Christopher C; Gao Wei; Wang Liqing; Busfield Samantha J; Wang Chichung; Qiu Yubin; Coyle Anthony J; Gutierrez-Ramos Jose-Carlos; Hancock Wayne W
Millennium Pharmaceuticals, Inc., Cambridge, MA 02139, USA.
Journal of experimental medicine (United States) Mar 18 2002, 195 (6) p795-800, ISSN 0022-1007 Journal Code: 2985109R
Contract/Grant No.: AI 40152; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
LIGHT (TNFSF14), a tumor necrosis factor superfamily member expressed by activated T cells, binds to herpes virus entry mediator (HVEM) which is constitutively expressed by T cells and costimulates T cell activation in a CD28-independent manner. Given interest in regulating the effector functions of T cells in vivo, we examined the role of LIGHT-HVEM costimulation in a murine cardiac allograft rejection model. Normal hearts lacked LIGHT or HVEM mRNA expression, but allografts showed strong expression of both genes from day 3 after transplant, and in situ hybridization and immunohistology-localized LIGHT and HVEM to infiltrating leukocytes. To test the importance of LIGHT expression on allograft survival, we generated LIGHT-/- mice by homologous recombination. The mean survival of fully major histocompatibility complex-mismatched vascularized cardiac allografts in LIGHT-/- mice (10 days, $P < 0.05$) or cyclosporine A (CsA)-treated LIGHT+/+ mice (10 days, $P < 0.05$) was only slightly prolonged compared with LIGHT+/+ mice (7 days). However, mean allograft survival in CsA-treated LIGHT-/- allograft recipients (30 days) was considerably enhanced ($P < 0.001$) compared with the 10 days of mean survival in either untreated LIGHT-/- mice or CsA-treated LIGHT+/+ controls. Molecular analyzes showed that the beneficial effects of targeting of LIGHT in CsA-treated recipients were accompanied by decreased intragraft expression of interferon (IFN)-gamma, plus IFN-gamma-induced chemokine, inducible protein-10, and its receptor, CXCR3. Treatment of LIGHT+/+ allograft recipients with HVEM-Ig plus CsA also enhanced mean allograft survival (21 days) versus wild-type controls receiving HVEM-Ig (mean of 7 days) or CsA alone ($P < 0.001$). Our data suggest that T cell to T cell-mediated LIGHT/HVEM-dependent costimulation is a significant component of the host response leading to cardiac allograft rejection.
Record Date Created: 20020319
Record Date Completed: 20020416
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DIALOG(R)File 155:MEDLINE(R)

09479987 21256041 PMID: 11356977
Novel, soluble isoform of the herpes simplex virus (HSV) receptor nectin1 (or PRR1-HIgR-HveC) modulates positively and negatively susceptibility to HSV infection.
Lopez M; Cocchi F; Avitabile E; Leclerc A; Adelaide J; Campadelli-Fiume G ; Dubreuil P
Institute of Cancer Biology and Immunology, Institut de la Sante et de la Recherche Medicale U.119, 13009 Marseille, France.
Journal of virology (United States) Jun 2001, 75 (12) p5684-91, ISSN 0022-538X Journal Code: 0113724
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A novel member of the nectin family, nectin1gamma, was molecularly cloned. The cDNA has the same ectodomain as nectin1alpha and nectin1beta, the two known transmembrane isoforms that serve as receptors for herpes simplex virus (HSV) entry into human cell lines (nectin1alpha and nectin1beta, also called PRR1-HveC and HIgR, respectively). The 1.4-kb transcript, which originated by alternative splicing, is expressed in human cell lines, and appears to have a narrow distribution in human tissues. The sequence does not have a hydrophobic anchoring region, and the protein is secreted in the culture medium of cells transfected with the cDNA. Nectin1gamma, purified from culture medium, can compete with membrane-bound nectin1beta and reduce HSV infectivity. The expression of nectin1gamma cDNA in cells resistant to HSV infection and lacking HSV receptors enables HSV to enter the cell, which implies that it is present at the cell surface. Thus, nectin1gamma has the potential both to mediate and to reduce HSV entry into cells.
Record Date Created: 20010517
Record Date Completed: 20010621
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DIALOG(R)File 155:MEDLINE(R)

11855640 99296730 PMID: 10366573
Functional characterization of the HveA homolog specified by African green monkey kidney cells with a herpes simplex virus expressing the green fluorescence protein.
Foster T P; Chouljenko V N; Kousoulas K G
School of Veterinary Medicine, Louisiana State University, Baton Rouge, Louisiana, 70803, USA.
Virology (UNITED STATES) Jun 5 1999, 258 (2) p365-74, ISSN 0042-6822 Journal Code: 0110674
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
We cloned the gene specified by African monkey kidney cells (Vero) that codes for the homolog of the herpes virus entry mediator (HveA) specified by HeLa cells. The primary sequence of the monkey HveA (HveAs) differed significantly from HveA. Single amino acid differences were distributed throughout the amino and carboxyl terminal portions of the HveAs in comparison with the HveA, whereas certain regions were highly conserved. The predicted membrane spanning domains of the two receptors differed substantially due to insertions and deletions of short amino acid sequences. The ability of HveAs to mediate HSV virus entry was tested in a series of experiments using the recombinant virus KOS/EGFP, which constitutively expressed the enhanced green fluorescence protein (EGFP) and Chinese hamster ovary cells (CHO) transformed with the HveAs gene. The KOS/EGFP virus was constructed by inserting an EGFP gene cassette within the intergenic region between the UL53 (gK) and UL54 (ICP27) genes. The KOS/EGFP virus formed viral plaques and replicated as well as the wild-type KOS virus. HveAs-transformed CHO cells constitutively expressing HveAs mediated herpesvirus entry efficiently, whereas cells transformed with the HveAs gene in the noncoding orientation did not mediate virus entry. A genetically engineered protein composed of the amino-terminal portion of the HveAs protein fused to the heavy chain of mouse IgG immunoglobulin as well as mouse antibodies raised against HveAs blocked virus entry into HveAs-transformed CHO cells. Thus, HveAs is the functional homolog of HveA.

Record Date Created: 19990706
Record Date Completed: 19990706

15/7/10
DIALOG(R)File 155:MEDLINE(R)

11846025 99286814 PMID: 10358762
Tumor necrosis factor receptor and Fas signaling mechanisms.
Wallach D; Varfolomeev E E; Malinin N L; Goltsev Y V; Kovalenko A V; Boldin M P
Department of Biological Chemistry, Weizmann Institute, Rehovot, Israel. lvwalach@weizmann.weizmann.ac.il
Annual review of immunology (UNITED STATES) 1999, 17 p331-67, ISSN 0732-0582 Journal Code: 8309206
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Four members of the tumor necrosis factor (TNF) ligand family, TNF-alpha, LT-alpha, LT-beta, and LIGHT, interact with four receptors of the TNF/nerve growth factor family, the p55 TNF receptor (CD120a), the p75 TNF receptor (CD120b), the lymphotoxin beta receptor (LT beta R), and herpes virus entry mediator (HVEM) to control a wide range of innate and adaptive immune response functions. Of these, the most thoroughly studied are cell death induction and regulation of the inflammatory process. Fas/Apo1 (CD95), a receptor of the TNF receptor family activated by a distinct ligand, induces death in cells through mechanisms shared with CD120a. The last four years have seen a proliferation in knowledge of the proteins participating in the signaling by the TNF system and CD95. The downstream signaling molecules identified so far--caspases, phospholipases, the three known mitogen activated protein (MAP) kinase pathways, and the NF-kappa B activation cascade--mediate the effects of other inducers as well. However, the molecules that initiate these signaling events, including the death domain- and TNF receptor associated factor (TRAF) domain-containing adapter proteins and the signaling enzymes associated with them, are largely unique to the TNF/nerve growth factor receptor family. (225 Refs.)
Record Date Created: 19990831
Record Date Completed: 19990831

15/7/11
DIALOG(R)File 155:MEDLINE(R)

11814276 99253915 PMID: 10318773
A newly identified member of tumor necrosis factor receptor superfamily (TR6) suppresses LIGHT-mediated apoptosis.
Yu K Y; Kwon B; Ni J; Zhai Y; Ebner R; Kwon B S
Department of Microbiology and Immunology and Walther Oncology Center, Indiana University School of Medicine and the Walther Cancer Institute, Indianapolis, Indiana 46202, USA.
Journal of biological chemistry (UNITED STATES) May 14 1999, 274 (20) p13733-6, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: AI28125; AI; NIAID; DE12156; DE; NIDCR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed

TR6 (decoy receptor 3 (DcR3)) is a new member of the tumor necrosis factor receptor (TNFR) family. TR6 mRNA is expressed in lung tissues and colon adenocarcinoma, SW480. In addition, the expression of TR6 mRNA was shown in the endothelial cell line and induced by phorbol 12-myristate 13-acetate/ionomycin in Jurkat T leukemia cells. The open reading frame of TR6 encodes 300 amino acids with a 29-residue signal sequence but no transmembrane region. Using histidine-tagged recombinant TR6, we screened soluble forms of TNF-ligand proteins with immunoprecipitation. Here, we demonstrate that TR6 specifically binds two cellular ligands, LIGHT (herpes virus entry mediator (HVEM)-L) and Fas ligand (FasL/CD95L). These bindings were confirmed with HEK 293 EBNA cells transfected with LIGHT cDNA by flow cytometry. TR6 inhibited LIGHT-induced cytotoxicity in HT29 cells. It has been shown that LIGHT triggers apoptosis of various tumor cells including HT29 cells that express both lymphotoxin beta receptor (LTbetaR) and HVEM/TR2 receptors. Our data suggest that TR6 inhibits the interactions of LIGHT with HVEM/TR2 and LTbetaR, thereby suppressing LIGHT- mediated HT29 cell death. Thus, TR6 may play a regulatory role for suppressing in FasL- and LIGHT-mediated cell death.

Record Date Created: 19990617

Record Date Completed: 19990617

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20may03 13:55:37 User208669 Session D2297.2

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$0.00 219 Type(s) in Format 6

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$10.36 Estimated cost File155

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$14.78 Estimated cost this search

$15.07 Estimated total session cost 2.730 DialUnits

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